

the kidney, and the elimination of the ammoniacal product is, in fact, the saving clause. It is when such persons take congestion of the lung, and are subjected to diminution of excretion by the lung, that the uræmic symptoms advance; and in these cases the breath is not ammoniacal during the attack. But there are other examples, where the uræmia is sudden in its appearance, owing to sudden arrest in the function of the kidney simply. Then the breath is markedly ammoniacal in the period of the acute attack. The third fact, as diagnostic of uræmic poisoning from poisoning by the ordinary narcotics, is, that during uræmic coma the patient will often rally and regain all his consciousness for a time, sinking again into forgetfulness, and even dying unconscious in the end.

The cause of death in uræmia formed the matter of another section of the paper, and was followed by observations on treatment. In the treatment of uræmic narcotism coming on suddenly, in a person not debilitated by previous disease, and not overloaded with fat, Dr. Richardson stated his belief that there was one ready and direct remedy, and that was free bloodletting. He had seen a man who had lain three days comatose and unconscious recover, under the immediate influence of loss of blood, so completely as to transact business affairs, and inquire into all that had occurred since he was struck down. Moreover, physiological reasons supported this treatment; for the bloodletting not only relieved the body from a portion of the poison, but removed the congestion of the kidney and of the other organs, and gave the permit for recovery, if recovery were possible. Thus, in animals in which artificial uræmia had been produced, the effect of frequent venesection tended greatly to prolong life. That bloodletting should not absolutely relieve in every case was reasonable, for whether relief were obtainable or not in any case would depend upon the degree of mechanical obstruction in the kidney; for if the obstruction were perfect, no treatment would be possible, seeing that no proceeding could be adopted to supplement the kidney altogether; but if in any instance there should be but partial obstruction, increased temporarily by congestion, then the act of abstraction of blood gave the only chance that remained of removing the burden from the excreting organ. The last part of the paper dwelt on uræmia in its forensic aspects. In many cases, where death is supposed to have occurred from the effects of small doses of opium or other narcotic, he (Dr. Richardson) believed that the cause was attributable to uræmia, and that so-called idiosyncrasies were probably intimately connected with renal disorder.—*Lancet*, Nov. 17, 1860.

17. *On Uræmia*.—Prof. JAKSCH holds that there are two varieties of uræmia which should be carefully distinguished; one being caused by the decomposition of urine and the absorption of carbonate of ammonia into the blood (ammonæmia), the other being the variety which accompanies Bright's disease of the kidneys. He has seen the former occur under the following circumstances: 1. In torpor and paralysis of the bladder; 2. In dilatation of the pelvis and calices of the kidney in consequence of the ureters being blocked up; and 3. In renal abscess, renal tuberculosis, and sacculated kidneys.

The following are the main differences characterizing the two forms of uræmia. We shall, to save circumlocution, use the word ammonæmia as the name of the one, and Bright's uræmia as the name of the other.

1. In advanced ammonæmia the urine discharged from the bladder manifests a strong ammoniacal odour, which Professor Jaksch has never noticed in any stage of Bright's uræmia. 2. Dropsical symptoms, either acute and febrile, or chronic and afebrile, have not been observed in ammonæmia. 3. Advanced ammonæmia is characterized by persistent dryness of the mucous membrane covering the mouth and fauces, as if every particle of moisture had been removed by blotting-paper; the membrane looks dry and shining, and the dryness even extends to the mucous membrane of the nose, the conjunctiva, and even to the chordæ vocales; these symptoms do not occur in Bright's uræmia. 4. The distinctly ammoniacal odour of the air exhaled, and of the cutaneous secretions of patients affected with ammonæmia, does not occur in Bright's disease. 5. Patients suffering from ammonæmia always show a marked dislike to meat, and especially brown meats, even if their affection has not advanced very far; a feature rarely seen in the other variety. 6. Professor Jaksch has never observed

in Bright's disease the violent intermittent rigors, simulating intermittent fever, which occur in ammonæmia. 7. In none of the cases of ammonæmia were convulsive or epileptiform attacks, nor croupy or diphtheritic exudations, noticed. 8. Disturbed vision, as produced in Bright's disease by exudation on the retina, does not appear to take place in ammonæmia. 9. Chronic ammonæmia is characterized by a uniformly pale and sallow complexion, and by gradually increasing emaciation; very acute and advanced ammonæmia is associated with very rapid wasting of the features, and muscular debility amounting to paralysis. 10. In all cases of ammonæmia which ran a rapid course there was vomiting, with concurrent or consequent diarrhœa; in chronic ammonæmia both phenomena were often entirely absent, or only occurred temporarily. 11. In ammonæmia, whether acute or chronic, Professor Jaksch has always seen death occur after sopor, varying in duration from several hours to several days.

The author of this valuable and interesting paper gives numerous cases illustrative of his views, and enters very fully into the various questions connected with diagnosis and treatment, for which we are unable to make room.—*Brit. and For. Med.-Chir. Rev.*, Oct. 1860, from *Vierteljahrsschrift für die praktische Heilkunde*, xvii., 1860.

18. *On the Resorption of Pleuritic Exudations.* By Prof. SKODA.—The resorption of pleuritic exudations frequently takes place very slowly, because the capillary vessels in the sub-pleural connective tissue are obliterated. This may be the result of shrivelling and disappearance of the connective tissue newly formed from the exudation, as then, in consequence of the arrest of the metamorphosis of tissue between the blood and the exudation, endosmose and exosmose cannot duly take place. It is not until after the lapse of months or years, when the fluid portion of the exudation has penetrated through the false membranes investing the pleura, that its resorption occurs. In the first case, internal medicines can, of course, avail nothing, as in them we possess no means of exciting the re-formation of vessels. What has been said explains the action of iodine injected in exudations, inasmuch as, by exciting inflammation, it causes the development of new vessels, and so induces the resorption of the effusion. But this view does not, perhaps, encourage us to the frequent employment of thoracentesis and subsequent injection of iodine; for this proceeding is by no means so safe as the corresponding operation for hydrocele. But, apart from that consideration, the injection of iodine or nitrate of silver into the pleural sac can answer no useful purpose; as, on the one hand, in consequence of the presence of the albuminous exudation, the caustic influence of these agents cannot reach the pleural sac, particularly as by the chemical combination which these substances form with the effusion, their power is altered and exhausted, so that the fluid must in the first instance be pumped out, which violent and sudden evacuation may be attended with evil consequences. But, on the other hand, in effusions of long standing, which have already attained to partial organization and shrivelling of the product of inflammation, the injection will be inefficacious, because the lung can now no longer fill the space previously occupied by the fluid exudation, especially as the investing false membranes must first be broken up by the lungs, which is not conceivable. But even if this should take place, a sudden evacuation could be followed by no favourable result, because necessarily there must be ruptures of the pleuritic adhesions and bursting of the compressed pulmonary parenchyma. Therefore, in a pleuritic effusion of long standing, it is only exceptionally that puncture is admissible, when the exudation is so considerable as to depress the diaphragm, to displace the mediastinum, and so to compress the lung that danger of suffocation supervenes. But how can the resorption of pleuritic exudations be induced? Experience shows that all those means which lower the pressure of the blood or augment the secretions, and therefore promote the separation of water from the blood, effect a diminution of the fluid effusion. Accordingly, venesections and diuretics may be indicated in cases of effusion; but these effects also occur spontaneously. In chronic exudations, which are already organized, such means will even be rather injurious, and the indication will be to employ remedies capable of dissolving solid exudations. Such remedies are iodine and mercury. The cautious employment of these